METHODS A systematic search of studies on the association of SNPs with susceptibility to CAD was conducted in PubMed, Embase, Cochrane Library and CNKI. Odds ratios (ORs) and 95% confidence intervals (95% CIs) were used to pool the effect size. A total of 6 case-control studies on rs28362491 in NFKB1-94ATTG were included.

RESULTS The significant association was found between rs28362491 polymorphism and CAD risk in four genetic models, (D versus I: OR = 1.10, 95% CI 1.04-1.18, $P_H = 0.509$; ID versus II: OR = 1.17, 95% CI 1.07-1.29, $P_H = 0.85$; DD versus II: OR = 1.18, 95% CI 1.04-1.34, $P_H = 0.465$; ID/DD versus II: OR = 1.18, 95% CI1.08-1.28, $P_H = 0.805$). A significant increased risk of CAD was observed in the rs28362491 polymorphism comparison, but there was insufficient data to fully confirm the association of CAD and rs28362491 in NFKB1-94ATTG.

CONCLUSIONS NFKB1-94ATTG ins/del rs28362491 polymorphism is correlated with CAD risk. However, the results of NFKB1-94ATTG rs28362491 should be interpreted with caution due to limited sample and heterogeneity. Large-scale and well designed studies are needed to validate our findings.

GW26-e2361

Association of APOB genetic polymorphisms and Aortic valve calcification in Han populations in Xinjiang, China

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OBJECTIVES Limited information is available when it comes to the impact of genetic on valvular calcification. Apolipoprotein B (apoB) is a key component in lipid metabolism and plays an important role in the dynamic equilibrium of cholesterol. The objective of this study was to investigate the association between aortic valve calcification and apoB genetic polymorphisms in the Han, Uygur and Kazak populations in China.

METHODS 583 participants, including 172 cases with aortic valve calcification and 411 controls were selected for the present study. Two SNPs (rs6725189 and rs693) of apoB were genotyped by using the polymerase chain reaction-restriction fragment length (PCR-RFLP) method. Independent-sample t-test, chi-square test and logistic regression were used to analyze.

RESULTS The rs6725189 was found to be associated with aortic valve calcification in the dominant model, and the difference remained statistically significant following multivariate adjustment ($p = 0.036$, $p = 0.004$, respectively). The rs693 was found to be associated with aortic valve calcification in the recessive model, and the difference remained statistically significant following multivariate adjustment ($p = 0.004$, $p = 0.028$, respectively).

CONCLUSIONS Both rs6725189 and rs693 of the apoB gene are associated with aortic valve calcification in the Han and Kazak populations of China.

GW26-e2444

MtDNA as a biomarkers in acute myocardial infarction and its effects on myocardial cell

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OBJECTIVES An increasing studies have focused on the phenomenon that mitochondrial DNA (mtDNA) activates innate immunity responses. However, the specific role of mtDNA in acute myocardial infarction remains elusive. This study was designed to examine whether mtDNA can be served as a biomarker of acute myocardial infarction (AMI) patients and try to eliminate the damage effects of mtDNA on cardiomyocyte.

METHODS Plasma nuclear and mtDNA levels were measured by quantitative PCR in 50 AMI patients, 50 non-myocardial infarction (MI) (with MI risk) and 50 healthy control. Purified mtDNA or nuclear DNA was added to H9c2s cells, with or without pretreatment with chloroquine (an inhibitor of endosomal receptors like TLR9). The cell viability and apoptosis were tested by MTT and Flow cytometry, respectively. The levels of TLR9, p-p38 mitogen-activated protein kinase (MAPK) and caspase 3 were detected by western blot.

RESULTS The concentrations of mtDNA were significantly higher in the AMI group of hospital day 1 than that in the non-MI controls and healthy individuals (3.754±0.384 ng/μL vs. 1.851±0.3483 ng/μL, $P<0.05$; 3.754±0.384 ng/μL vs. 0.1517±0.0924 ng/μL, $p<0.05$) and decreased shortly after PCI. Exogenous mtDNA reduced the viability of H9c2 cells and induced TLR9 and p-p38 MAPK and caspase 3 activation. These effects were inhibited by chloroquine. Nuclear DNA did not induce TLR9, caspase3, p-p38 MAPK activation.

CONCLUSIONS MtDNA level is increased after AMI and can used as a biomarker in AMI patients. MtDNA activates TLR9-P38MAPK and inducing cardiomyocyte cells death.

GW26-e2445

Protective effects and its mechanism of Helix B-surface peptide against cardiac microvascular endothelial cell injury induced by ischemia / reperfusion

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OBJECTIVES MI/R injury could paradoxically reduce the beneficial effects of myocardial reperfusion and cause contractile dysfunction and cellular damage, lacking of effective strategies of prevention cure. A helix-B surface peptide (HBSP), which is composed of 11 amino acids derived from the aqueous face of helix B of EPO, recently was developed and retained tissue-protective but avoiding erythropoietic property of EPO. Our previous experiment results have demonstrated that HBSP could reduce myocardial ischemia / reperfusion (MI/R) injury of Sprague-Dawley rats and increase the post ischemic myocardial functions via activating the phosphatidylinositol 3-kinase (PI3-K)-Akt cascade. However, it is still unclear whether HBSP can protect the cardiac microvascular endothelial cells (CMECs) when subjected to ischemia / reperfusion injury Therefore, the aims of the present study were to investigate the protective effect of HBSP against cardiac microvascular endothelial cells (CMECs) injury induced by ischemia / reperfusion and further explored the underlying mechanisms involved.

METHODS CMECs isolated from the adult hearts of Sprague-Dawley rats were exposed to hypoxia and ischemia buffer for 2h followed by 4h reoxygenation. Then CMECs were randomized to receive different concentrations of EPO-derived peptides HBSP, EPO, HBSP plus LY294002 (specific inhibitor of PI3K), HBSP plus rapamycin (specific inhibitor of mTOR) at the start of reperfusion. The cell viability of CEMCs was measured by MTT colorimetric assay and the apoptosis of CEMCs was detected by Tunel method. The wound scratch assay and transwell method were performed to detect the migration of CMECs. The expression of p-AKT, p-mTOR and p-p70S6K were analyzed by western blot analysis.

RESULTS Both cell viability and migration ability of CMECs were impaired after SI/R ($P<0.01$ vs.control), and the apoptotic index increased in comparison with control group ($P<0.01$). While administration of EPO and HBSP during reperfusion dramatically attenuated the dysfunction of CMECs. Compared with the SI/R group, HBSP treatment in CMECs exerted protective effects as evidenced by the increase of cell viability ($P<0.05$), inhibited CMECs apoptosis (25.5%± 0.43% vs. 41.1%±0.8%, $P<0.01$) and improved the migration ability of CMECs ($P<0.05$). Moreover, HBSP caused over Akt phosphorylation in the reperfusion CMECs, which was abrogated by the treatment of LY294002 ($P<0.05$), but not by rapamycin. Furthermore, mTOR phosphorylation following HBSP treatment was prevented by either LY294002 or rapamycin ($P<0.05$). Similarly, the phosphorylation of the mTOR downstream molecule p70S6K were up-regulated by HBSP treatment ($P<0.05$). While treating with LY294002 or rapamycin prevented HBSP-induced phosphorylation of p70S6K ($P<0.05$). Compared with the HBSP group, the apoptotic index increased while treating with LY294002 or rapamycin ($P<0.05$).

CONCLUSIONS HBSP might have protective effect of CMECs against ischemia/reperfusion injury, which may be related with activation of PI3K/AKT/mTOR signaling pathway.

GW26-e2449

The Role of Calpain in Myocardial Apoptosis Induced by Oxidative Stress in Mouse cardiomyocytes Hypoxia/Reoxygenation

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OBJECTIVES In the present study, we aimed to explore the effects of calpain and its inhibitor PD150606 (PD) on oxidative stress induced myocardial apoptosis in mouse hypoxia/reoxygenation (H/R) injury.

METHODS The ventricular myocytes of adult C57BL/6 mice were isolated and cultured. The cardiomyocytes were randomly divided